REMARKS

Favorable reconsideration is respectfully requested in view of the foregoing amendments and the following remarks.

I. CLAIM STATUS AND AMENDMENTS

Claims 4-9 were pending in this application when last examined.

Claims 4-9 were examined on the merits and stand rejected.

Claims 4-6 are cancelled without prejudice or disclaimer thereto.

II. OBVIOUSNESS REJECTION

On pages 3-7 of the Office Action, claims 4-9 were rejected under 35 U.S.C. 103(a) as obvious over Spiegelman et al., Vega et al. /Vega et al. (Abstract) and Saldek et al.

Applicants respectfully traverse this rejection as applied to the remaining claims.

The invention of claim 7 is a method for screening for compounds that increase the activity of ERR for expressing MCAD gene. The basic principles of this method are:

(1) resistance to obesity or diabetes can be maintained by controlling energy balance in the body by regulating fatty acids β-oxidation by MCAD gene expression

(2) MCAD gene expression is regulated by ERR

(3) transcriptional activity of ERR is activated by binding with its ligand, ERRL1.

The specification notes that a relationship between ERRL l, ERR and MCAD was known. Please see page 2, line 29 to page 3, line 3 of the specification. However, it was not previously confirmed that MCAD gene expression for controlling energy balance would be precisely regulated by ERR *in vivo*. This is because an *in vivo* substance to regulate ERR was not known. In this regard, the Examiner alleges that the relationship between ERR and ERRL1 is known from Vega and Saldek. For example, the Examiner states, "At the time of the invention,

ERRL1 (PGC-1) was known to induce MCAD gene" (see page 6, lines 20-21 of Office Action). Please note, however, that ERRLl was previously named "PGC-2", which is different from "PGC-1". In addition, the Examiner stands on the relationship between PGC-1 and PPAR alpha. That is, while the Examiner's speculation is based on the relationship between PGC-1, PPAR

and MCAD, the principle of this invention is a relationship between ERRL1 (PGC-2), ERR and MCAD.

In particular, Figure 1 of the specification lists both ERRL1 and PGC-1. Such sequences, as shown in the figure, are different. Thus, the teachings of Vega and Saldek do not teach or suggest the relationship of ERRL1 (PGC-2), ERR and MCAD.

Applicants further note that on pages 30-34 of the specification, it is shown that increased expression of ERRL1 results in precise control of energy balance. In particular, transgenic mice over-expressing ERRL1 were hyperphagic but lean. Such results confirm that *in vivo* ERRL1 controls energy balance. Thus, an assay comparing MCAD expression caused by a candidate compound versus that caused by ERRL1 is useful for screening for compounds that increase the activity of ERR for expressing the MCAD gene.

It is therefore noted that the cited references fail to teach or suggest the relationship between ERRL1, ERR and MCAD. Further, the cited references fail to teach or suggest *in vivo* activity of ERRL1. Thus, the cited references fail to teach or suggest the claimed screening method using ERRL1.

Thus, for the above noted reasons this rejection is untenable and should be withdrawn.

CONCLUSION

In view of the foregoing amendments and remarks, it is respectfully submitted that the present application is in condition for allowance and early notice to that effect is hereby requested.

If the Examiner has any comments or proposals for expediting prosecution, please contact the undersigned attorney at the telephone number below.

Respectfully submitted,

Akira KAKIZUKA et al.

/William R. Schmidt, II/

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